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THE EIGHTH ANNUAL CHARLES V. CHAPIN ORATION*

THE CONTROL OF PELLAGRA

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I WISH to express my deep appreciation to the Rhode Island Medical Society for the high honor conferred on me by asking me to deliver the Chapin Memorial Lecture for 1949. Dr. Chapin suggested so many of the practical measures upon which modern public health is based that I am sure his scientific mind would grasp quickly the technical problems which faced us in the beginning. It has been the aim of our group, as it was Dr. Chapin's, to get at the facts of the situation, to seek the best known scientific information at the time, to seek other relevant information about the unsolved parts of the problem, and to put this information together so that we and other physicians then could apply measures for the good of all.

Let us think for a moment of pellagra. The authentic history of the disease began in northern Spain in 1735 when Gaspar Casal, physician to King Phillip V, recorded his observations on "mal de la rosa," an illness prevalent among the peasants in the province of Asturias and later shown to be the same disease as pellagra. Twenty years after Casal recognized the disease in Spain it was recognized in northern Italy but the term pellagra, derived from the Italian "pelle agra," meaning rough skin, was not used until 1771 by the Italian physician Frapoli, who found the word in common use among the peasants of Lombardy. Subsequently, numerous cases were recognized throughout northern and central Italy and a special hospital for the treatment of the disease was established at Legano in 1784 by a warrant of Joseph II of Austria. As in Spain and Italy, the disease already was widespread at the time of its recognition in many countries, chiefly France, Egypt, and Roumania.

The first mention of the disease among English-speaking peoples appeared in 1890 in Surgeon Billings' *National Medical Dictionary*, in which pellagra was defined as "an endemic disease of Italy characterized by chronic erythematous inflammation of the skin with digestive derangement and neuroses." Five pages on pellagra, much of it devoted to the thesis that the disease undoubtedly was caused by a diet of "bad maize," appeared in *The System Of Medicine* edited by Clifford Albutt and published in 1900. It was in this publication that the term "pellagra sine pellagra" first was encountered. In the United States the first cases of the disease were reported by Dr. Gray in Utica, New York and Dr. Tyler in Somerville, Massachusetts.

Early American Studies

In 1907, Dr. George Searcy and Dr. E. L. McCafferty, physicians on the staff of the Institution for Insane Negroes at Mount Vernon, Alabama, startled the country when they published a brief report of an epidemic of pellagra in this institution.

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These studies have been aided by many philanthropic persons, but during the most recent past the continuation of studies has been made possible by grants from Hoffman-LaRoche, Inc.

So accurate was their description of the disease that within a few weeks after their report was published, pellagra was recognized in six states and its identity with Italian pellagra was irrevocably established. Goldberger, Wheeler, and their associates[1], a decade or so later, induced pellagra in 5 of 11 subjects by giving them an unbalanced diet. Later, Goldberger and Tanner demonstrated the pellagra-preventive value of dried brewers' yeast, lean meat, and milk[2] and they treated some cases of naturally-occurring deficiency diseases with dried brewers' yeast[3]. Despite the application of therapy, Boggs and Padget[4] and Spies and De Wolf[5] reported a high mortality rate (over 50 per cent) among severely ill pellagrins in the early 1930's. Moreover, uncounted thousands of pellagrins were committed to custodial institutions for the legally insane.

After observing the high death rate in severe pellagra, I looked through the literature to see how many remedies had been recommended and I found over two hundred. This made me feel that none of them were specific. I then tried to find the criteria used for determining the effectiveness of a recommended therapeutic agent and found that usually they were subjective, not objective. Frequently, the effectiveness of an agent was judged by the disappearance of the skin lesions of pellagra. I then put a group of pellagrins on a pellagra-producing diet to try to find an objective method of measuring the effectiveness of a potential therapeutic agent[6]. I learned that the dermal lesions disappeared while the patients were restricted to a pellagra-producing diet but that frequently the patients developed a sore mouth and tongue and lost a great deal of body weight. Thus, restricting patients to a pellagra-producing diet gave us a way of studying the tongue lesions, which proved to be an accurate index for measuring the potency of a potential anti-pellagra remedy. This diet was studied still further[7] and we found that we could produce what was called "rat pellagra" on the same diet on which the pellagrous skin lesions in human beings disappeared. This demonstrated that what was called "rat pellagra" was not the same as human pellagra, a finding that has been amply confirmed many times since.

The Use of Yeast

It occurred to me that yeast in rather small amounts might prevent the disease and still not cure it and that perhaps the quantity of yeast required to cure it was much larger than the amount needed to prevent it. To test this hypothesis, my associates and I selected a group of severely ill pellagrins and admitted them to the hospital[8]. We soon learned that instead of giving 1 ounce of dried brewers' yeast powder daily, it was wise to give 6, 8, or even 10 ounces daily. Also, we found that many pellagrins had coexisting diseases and by treating these diseases the patients often were kept alive. In some instances, however, the patients were so severely ill they could eat only with difficulty because of severe stomach distress. Frequently, they had vomiting and intractable diarrhea. In these cases we realized the advisability of injecting some therapeutic material whenever possible. After many preliminary observations, we found that these cases could be benefited by large injections of liver extract. By these methods of therapy the death rate fell from 54 to 6 percent and we were convinced that the former frequent failure to respond to therapy had been due to the inadequate amounts of the therapeutic agents administered and failure to treat coexisting diseases. In 1931 we began a study of the relationship of chronic alcoholic addiction to pellagra[5]. A large group of patients with so-called alcoholic pellagra were admitted to the hospital with the understanding they would be given all the whiskey they wished to drink provided that we measured it and that they ate all they were given of a liberal, well-balanced diet, yeast, or liver. They soon learned that if they did not eat they would get no whiskey so they ate huge amounts of food, drank heavily, and were happy. Despite the fact that they continued to drink between a quart and a quart and a half of whiskey daily, their symptoms of pellagra did not become worse if they continued treatment. At this time we advanced the hypothesis that so-called alcoholic pellagra was a form of true pellagra. To test this hypothesis, 50 cases of severe endemic pellagra were selected in Birmingham, Alabama, an area in which pellagra was endemic and in which the death rate was high, irrespective of the method of treatment applied[9]. In these 50 cases we applied the therapeutic methods that we had used in the treatment of the cases of so-called alcoholic pellagra. Three patients died, and at post mortem all revealed adequate explanation for death aside from the residual lesions of pellagra (the acute manifestations had disappeared prior to death). The remainder recovered. Since alcoholic and endemic pellagra appeared identical and responded similarly to identical therapy, we became convinced that the two were the same disease.

Nicotinic Acid Effective

Soon after this, students of pellagra became interested in nicotinic acid. We previously had found that liver extract when injected in very large amounts to severely ill pellagrins might be life-saving[10]. We found also that liver extract was effective in many patients whose mouths were so sore they could not eat but that in these cases it was necessary to inject massive doses. Goldberger and Sebrell established the value of liver extract in treating canine blacktongue[11]. Elvehjem, Mad-

den, Strong, and Woolley showed that nicotinic acid was curative in canine blacktongue and isolated it from liver extract[12]. Independently, and almost simultaneously, excellent results in treating human pellegra were reported by our group[13], by Fouts, Lepkovsky, Helmer, and Jukes[14], by Smith, Ruffin, and Smith[15], and by Harris[16].

Mental changes as a part of the pellagra syndrome had long been recognized by physicians in areas where pellagra was endemic. Frequently, however, they occurred before other symptoms appeared or in the absence of other diagnostic criteria. Pellagrins with mental changes often were committed to institutions for the legally insane. In 1938 my associates and I showed that nicotinic acid in adequate amounts was specific for the acute mental symptoms of pellagra and that frequently, large repeated doses of nicotinic acid prevented the development of mental symptoms in subclinical and mild pellagra[17].

After finding nicotinic acid to be effective in treating pellagra and in preventing recurrences under hospital conditions, we thought it would be worthwhile to study its effect on pellagrins living at home without change in environment or activities[18]. A large group of patients who were subject to one or more recurrences of pellagra each year were selected for study. Within from one to three days after treatment with nicotinic acid was initiated, nearly all the patients volunteered that they felt much better and the dizziness, depression, burning sensations, confusion, and "upset head" had disappeared.

The discovery of the specificity of nicotinic acid for pellagra led to the recognition of mixed vitamin deficiencies and their treatment. Studies of a large series of cases showed that pellagrins were prone to develop multiple deficiencies. When adequate amounts of nicotinic acid were administered to pellagrins, the mucous membrane lesions, alimentary tract, mental symptoms, and erythematous dermal lesions improved rapidly but if these patients continued to eat their usual deficient diets, beriberi and riboflavin deficiency frequently developed[19]. This was not surprising since detailed dietary studies of the foods consumed by pellagrins revealed inadequacies of most of the essential nutrients. Thus, we came to consider pellagrins as having nutritive failure in the same sense that you might call heart failure myocardial failure. The term "nutritive failure" perhaps means different things to different physicians. It is used in our Clinic to describe a variable clinical picture and does not have a specific physiological connotation. It does not indicate why the nutrition has failed but simply that it has failed. Our work has convinced us that many times the physician cannot determine at once the physiologic basis of nutritional insufficiency, and by the use of this term he can describe briefly the clinical picture without knowing the precise cause. The term has become so commonly entrenched in our clinical usage that it does not seem wise for us to restrict it or to change it. For the sake of clarity, the various disturbances and mechanisms which create a picture of nutritive failure, we often describe separately. We can see pellagrous dermatitis or glossitis, beriberi neuritis or beriberi heart disease, scorbutic hemorrhages of gingivitis, cheilosis or the ocular symptoms of riboflavin deficiency as present symptoms arising as a response of the body to a long-continued deprivation of certain nutrients in food. Hence, nutritive failure is a more inclusive term than pellagra, beriberi, scurvy, or riboflavin deficiency in that it connotes varying degrees of mixed deficiencies operating simultaneously.

I think it is extremely important to realize that in all the scientific literature one cannot find a single report of failure in treating pellagra since 1938. In our Clinic we have not seen a single case in three years, whereas, we used to have 10,000 in a single year. With an infinitesimal amount of money these people can be cured and can go back to work. Most of the pellagrins who used to die were paupers who had to be buried by the state, and that was expensive. Five per cent or so of the persons who filled the asylums were pellagrins, particularly in the South, and they had to be cared for the rest of their lives. Where you have big institutions supported by taxes, you have bureaucracies; and the more people supported in that kind of institution, the more bureaucracies we may have in time. The more specific remedies that can be worked out, the less danger there is of having these things. To me, this is the best approach toward preventing any part of socialized medicine.

Nutritional Macrocytic Anemia

Nutritional macrocytic anemia often accompanies pellagra[20]. In this type of anemia the bone marrow and peripheral blood findings are similar to those in pernicious anemia. When folic acid is administered to persons with nutritional macrocytic anemia, the reticulocytes (young red blood cells) begin increasing[21]. Many patients with nutritional macrocytic anemia have severe diarrhea. Sometimes one bowel movement will fill a quart jar. On the fifth day after folic acid therapy is initiated, the stools usually begin to return to normal. When the diarrhea begins to subside, the patients frequently complain that they are constipated. They are not constipated but they have had diarrhea for so long that they do not realize what a normal bowel movement is. X-rays of the gastrointestinal tract of these patients show spasms of the alimentary tract. Following folic acid therapy, alimentary tract function returns to normal.

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Soon after folic acid was found to be effective in producing a hemopoietic response in several types of macrocytic anemia in relapse, we found that 5-methyl uracil, an entirely different chemical compound, produced a similar effect in persons with these diseases[22]. Despite the fact that these substances produced a blood response in pernicious anemia however, they did not cure the nerve degeneration which so frequently develops in persons with this disease; neither did they prevent or cause it. Folic acid and 5-methyl uracil (thymine) are incomplete treatments for pernicious anemia in that they do not protect the nervous system.

The finding of the effect of specific therapeutic agents of known chemical composition on the cells of the bone marrow has opened a fresh and fertile field for the clinical investigator. While we were studying and trying to redefine the macrocytic anemias in the light of all the various loose threads which enter the meshwork of their pathogenesis, we steadily were observing that a large amount of these substances was necessary to produce a satisfactory hemopoietic response. When we considered that relatively little highly potent liver extract was needed, we naturally were led to think that other substances are contained in liver extract.

The most recently discovered antianemic substance is vitamin B_{12}, a chemical compound of unknown chemical structure, which was isolated from liver[23], [24], and found to be effective in treating pernicious anemia[25], [26], nutritional macrocytic anemia[27], and tropical sprue[28], [27], diseases which often are associated with pellagra. When vitamin B_{12} is administered to patients with these diseases, reticulocytosis occurs and is followed by an increase in red blood cells, platelets, and hemoglobin. A striking clinical response parallels the hemopoietic response. About the time the reticulocytes begin to rise, the patients volunteer that they feel stronger. Those whose appetites have failed have a sudden desire for food. Symptoms arising from subacute combined degeneration of the spinal cord in persons with pernicious anemia are decreased by the administration of vitamin B_{12}. The severe glossitis present in some cases of macrocytic anemia heals spectacularly. In patients with sprue and nutritional macrocytic anemia, there is a decrease in the number of stools and a tendency for the stools to return to normal.

Vitamin B_{12}, per unit of weight, is the most effective antianemic substance known. It has been found to produce a hematologic response in persons who have pernicious anemia, nutritional macrocytic anemia, and tropical sprue. It is the only pure chemical substance known to be effective in relieving subacute combined degeneration of the spinal cord in persons with pernicious anemia.

Principles of Treatment

In this oration I have made an effort to describe the scientific basis, worked out in the last twenty years, for effecting improvement and rehabilitation in persons with pellagra and associated conditions. The pellagrin, like ourselves, is dependent on rapid response to changes in his external environment. These adjustments can be brought about as long as his nutrition permits, but if the deprivation of nutrients becomes sufficiently protracted, these protective mechanisms eventually fail. It has been clear for some time that the nutrients play an important role in the general physiologic processes. The great problem for the physician is to replenish the tissues, and clinical experience with the vitamins must continue for, as yet, we know little as to the restoration of the impoverished cells of the body. From the practical point of view, my own position is clear; that is, large doses should be administered and they should be prescribed by mouth unless for some reason parenteral administration is necessary. Vitamin therapy alone is not sufficient, and every time I lecture I stress that vitamins cannot take the place of a well-balanced diet. The whole matter of diet must be explained to the patient, and education in this regard is time-consuming. The principles of treatment which we used in rehabilitating persons with pellagra and other vitamin deficiency diseases are as follows:

1. Conditions causing excessive requirements for nutrients were removed or relieved whenever possible.
2. Symptomatic treatment and treatment for coexisting diseases were given.
3. It was made certain that the patient ate daily a diet which supplied 3000 to 4000 calories, 120 to 150 grams of protein, and liberal amounts of minerals and vitamins.
4. Therapeutic substances, such as dried brewers' yeast powder, liver extract, or vitamins in the form of synthetic substances, were administered in sufficient amounts to correct the deficiency.

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Dr. Tom D. Spies of Birmingham, Alabama, receives the Dr. Charles V. Chapin Medal of the City of Providence from Councilman Howard Presel, chairman of the City Council award committee, at the annual dinner of the Rhode Island Medical Society, at Providence, May 11, 1949

LYMPHOSARCOMA AND HODGKIN'S DISEASE*

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THE SO-CALLED LYMPHOBLASTOMA or malignant lymphoma group of cases generally includes leukemia, lymphosarcoma and Hodgkin's Disease. Because of the complexities and relationships of the leukemia group and because they are always generalized diseases and seemingly incurable by present methods of treatment, they will be omitted in this presentation.

The important feature about both lymphosarcoma and Hodgkin's Disease is that some cases are apparently focal in origin and hence potentially curable if the focus can be totally removed or completely destroyed before it has spread to other parts of the body. The word focal has been qualified by "apparently" because some doubt exists as to whether or not this is the truth and the evidence for and against this proposition will be presented.

But first it will be well to provide some general information about the two diseases so that the problem may be better understood.

Lymphosarcoma, as its name implies, is a tumor composed of immature cells of the lymphatic tissues, specifically the lymphoblasts and reticulum cells. While 75 per cent of all cases appear first in the lymph nodes, the other quarter manifest themselves elsewhere especially in the gastrointestinal tract, the upper respiratory and alimentary tracts including the salivary glands and in the skin, bone marrow, and orbit. Only sporadic cases are found starting in other regions. Occasionally such manifestations appear to be truly focal. When these tumors start to proliferate they reproduce masses of lymphoblastic cells which have more or less resemblance to immature lymphoblasts and reticuloblasts. Because exact criteria for recognizing these cells have not been established various names have been applied to the tumors by different authors. This has resulted in some confusion. Clinically this is not very important. The majority call the small cell tumors lymphocytic cell lymphosarcomas and the large cell ones reticulum cell lymphosarcomas but actually there is no important difference between the two in degree of malignancy, radiosensitivity or in any other way. There is a third variety which is worth recording separately because it shows some degree of differentiation. In the lymphocytic and reticulum cell forms the entire lymph node is replaced by solid masses of the tumor cells, but in this third variety some degree of differentiation is maintained by the formation of giant distorted follicles. This variant was thought at first to be a queer form of lymphoid hyperplasia but there is good evidence that it is a true neoplasm albeit a somewhat less malignant one with a slower development and a more protracted course. If the giant follicles are well formed and have clearly defined margins they are much less malignant than if the follicles are poorly defined and merge imperceptibly with the surrounding nodal cells. In this study, the lymphosarcomas will be divided into lymphocytic cell, reticulum cell and giant follicle forms and the giant follicle tumors will be further subdivided into well and poorly differentiated tumors.

These varieties may change their types but if they do it is always for the worse and never for the better. Thus a well differentiated giant follicle tumor may eventually assume the characteristics and malignancy of the reticulum or lymphocytic cell tumors and all three varieties may terminate as leukemia. It has been stated that the giant follicle form can terminate as Hodgkin's disease. It is improbable that this is true. In this writer's opinion the error has resulted from a failure to recognize the paragranulomatous form of Hodgkin's disease. It is possible for Hodgkin's disease to terminate as a reticulum cell sarcoma but the reverse is probably never true.

While *Hodgkin's Disease* in some ways resembles lymphosarcoma in that commonly it involves the lymphoid tissues of the body especially the lymph nodes, it differs because it is very rarely focal in origin and because microscopically it is granulomatous in appearance being compounded of

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a variety of inflammatory and reparative cells and very rarely of cells that are tumorous in appearance unless one can call the Reed-Sternberg cells neoplastic. While in about three-fourths of the cases the disease first manifests itself in the cervical nodes, no one can say whether or not this is its real site of origin and it may involve any or all of the lymph nodes as well as the spleen, liver, lungs, bone marrow, skin, thymus, alimentary tract, tonsils and breast. The first change is presumed to be a proliferation of reticulum cells. This is quickly followed by a disappearance of the follicles and sinuses which characterize organized lymphoid tissue, by the appearance of the bizarre small multinucleated cells called Reed-Sternberg cells and by patchy fibrosis and eosinophilic leucocytic cell infiltration. Rarely foci of necrosis develop. Jackson and Parker have recorded three subdivisions of Hodgkin's disease based upon certain histological appearances. The great majority they classify as Hodgkin's granuloma. Where the reticulum cells predominate and are markedly anaplastic, they suggest that the process has become a true lymphosarcoma which they characterize by the name Hodgkin's sarcoma. If the process is confined to cervical nodes on one side of the neck and only a part of the node appears involved chiefly by the proliferation of reticulum cells and other slight changes, they use the term Hodgkin's paragranuloma and expect the process to progress very slowly. In every instance, however, a diagnosis of Hodgkin's disease cannot be made unless true Reed-Sternberg cells are found. Since during life the diagnosis can only be made with assurance by biopsy, subdivision into these three groups cannot be done with certainty for it is well known that appearances can differ in different nodes and tissues of the same individual.

In spite of the interesting and sometimes brilliant palliative results which have been obtained in advanced and generalized cases of these diseases, and particularly Hodgkin's disease, by treatment with nitrogen mustard, urethane and other related substances, they have been in use too short a time for any one to know whether or not cures can be achieved by them. One must still turn to the results obtained by treatment with radiotherapy and surgery over long periods of time and compare them with the life histories of patients who have received no curative therapy.

Table I deals with 170 cases of lymphosarcoma seen at the Presbyterian Hospital, N. Y. during the 20 year period 1915-1934 inclusive. When it was prepared more than 10 years had passed subsequent to the end of 1934 so that all of these patients had had an opportunity to live 10 years or more. 119 of them had been treated by radiotherapy, surgery or a combination of the two, and 51 had received no curative treatment. This table shows that only one untreated patient survived more than five years and none 10 years. By contrast 28 treated patients lived five years, 16 ten years and all but one of the 16 were symptom free at last report. When the primary site of the long survivors is observed, it can be seen that proportionately very few were primary in lymph nodes and that better results on a percentage basis were attained when the lesion started elsewhere. The results here recorded for the lymphosarcomas pri-

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TABLE I

Lymphosarcoma (1915—1934 inclusive)

Primary Site	*All Cases* No. Cases	Survived 5+ Years No.	%	Symptom Free 5+ Years No.	%	Survived 10+ Years No.	%	Symptom Free 10+ Years No.	%	*Treated Cases* No. Cases	Survived 5+ Years No.	%	Symptom Free 5+ Years No.	%	Survived 10+ Years No.	%	Symptom Free 10+ Years No.	%
Cervical	34	4	11.8	2	5.9	2	5.9	2	5.9	26	3	11.15	2	7.7	2	7.7	2	7.7
Axillary	10	2	20	1	10	1	10	1	10	8	2	25	1	12.5	1	12.5	1	12.5
Fem., Ing.	18	5	28	2	11	3	17	2	11	14	5	35.7	2	14.3	3	21.4	2	14.3
Retroperit. Mesenteric	24	2	8.3	1	4.1	1	4.1	1	4.1	13	2	15.4	1	7.7	1	7.7	1	7.7
Mediastinal	8	0	0	0	0	0	0	0	0	3	0	0	0	0	0	0	0	0
Generalized	34	2	6	0	0	0	0	0	0	19	2	10.5	0	0	0	0	0	0
All Nodes	128	15	11.7	6	4.7	7	5.5	6	4.7	83	14	16.9	6	7.2	7	8.5	6	7.2
Mouth. Nasoph. Salivary Glds.	19	7	37	5	26.3	5	26.3	5	26.3	19	7	37	5	26.3	5	26.3	5	26.3
Gastrointest.	13	4	30.7	4	30.7	4	30.7	4	30.7	10	4	40	4	40	4	40	4	40
Skin. Orbit	9	3	33	0	0	0	0	0	0	6	3	50	0	0	0	0	0	0
Spleen	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
All Cases	170	29	17.1	15	8.8	16	9.5	15	8.8	119	28	23.5	15	12.7	16	13.4	15	12.7
										Untreated Cases								
										51	1	2	0	0	0	0	0	0

Reproduced from Stout, A. P., N. Y. State J. Med. 47:158-164, 1947.

mary in the skin and orbit are no doubt misleading because of lack of follow-up data. McGavic's figures for the lids and orbit indicate a 5 year cure rate of 50 per cent. This would suggest a 25 per cent cure rate at 10 years.

Table II shows the relative 5 and 10 year cure rates when the tumors are segregated according to type. The numbers are so small as to have little statistical significance but the indications are that there is litle important difference between reticulum and lymphocytic cell types but that the giant follicle

TABLE II

Results of Treatment
According to Type of Lymphosarcoma

Tumor Type	No. Cases	Survived 5 Years No.	%	Symptom Free 5 Years No.	%	Survived 10 Years No.	%	10 Years Symptom Free No.	%
All Cases									
Reticulum cell	78	12	15.4	6	7.7	7	9	6	7.7
Lymphocytic cell	40	8	20	3	7.5	3	7.5	3	7.5
Giant follicle	19	7	36.8	3	15.8	3	15.8	3	15.8
Treated Cases									
Reticulum cell	48	11	23	6	12.5	7	14.6	6	12.5
Lymphocytic cell	30	7	23.3	2	6.7	2	6.7	2	6.7
Giant follicle	16	7	43.8	3	18.8	3	18.8	3	18.8

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tumor seemingly has a better prognosis. Because of the small numbers in this group, Table III was prepared showing additional cases all of which had had an opportunity to live five or more years. This shows a total five year cure rate of 35.5 per cent. When these cases are further subdivided into completely and incompletely differentiated forms we find half of the completely differentiated and a quarter of the incompletely differentiated cases

TABLE III

Results of Treatment of 31 Patients with Giant Follicle Lymphosarcoma Treated More Than 5 Years Ago

Differentiation	No.	Died less than 5 yrs.	Died or alive with tumor 5+ yrs.	Symptom-free over 5 years
Complete	12	3 (25%)	3 (25%)	6 (50%)
Incomplete	19	11 (57.9%)	3 (15.8%)	5 (26.3%)
Total	31	14 (45.2%)	16 (19.3%)	11 (35.5%)

Reproduced from Stout, A. P.,
N. Y. State J. Med. 47:158-164, 1947.

surviving symptom-free for five years. In both instances these results are much better than for the other two varieties of lymphosarcoma. Finally one may ask, are these long survival treated lymphosarcoma cases really cured? Table IV shows the extent of our knowledge about this for it gives the latest available information about 16 long survival cases. Exclusive of one who died in the 11th year following treatment of generalized lymphosarcomatosis, the other 15 had no evidence of disease at last reports, three of them are dead in the 14th, 17th, and 18th years after treatment without evidence of lymphosarcoma and the other 12 were alive for varying periods up to over 17 years after treatment. Table V shows our latest data on long survival lymphosarcomas. There are 25 such individuals; 13 had primary manifestations in lymph nodes, 6 in the mouth and nasopharynx and 6 in the gastrointestinal tract. Twenty were alive and without evidence of lymphosarcoma, 3 others died without evidence of tumor. In these two groups 8 patients had passed the 15 year mark. Only two patients are known to have had reappearance of lymphosarcoma both in the 11th year after treatment when they died. One cannot give an absolute assurance that these 23

TABLE IV

16 out of 119 cases of lymphosarcoma treated during the years
1915-1934 inclusive and known to have survived 10 or more years

Sex	Age	Primary site	Tumor type	Treatment R.	S.	RS.	Result
M	50	Inguinal nodes	Reticulum		+		Died 10 years generalized.
M	69	Inguinal nodes	Lymphocytic	+			Well 10 years (case lost).
F	40	Inguinal nodes	Lymphocytic	+			Well 17 years.
M	39	Retroperitoneal nodes	Lymphocytic	+			Well 16 years.
F	58	Axillary nodes	Reticulum		+		Well 13 years 5 mos. (case lost).
M	20	Cervical nodes	Giant follicle	+			Died 16 years 1 mo. No recurrence.
M	13	Cervical nodes	Giant follicle			+	Well 13 years.
F	62	Tonsil nodes	Reticulum			+	Well 16 years 6 mos.
F	62	Tonsil nodes	Reticulum	+			Died 13 years 8 mos. No recurrence.
F	20	Buccal mu.	Lymphocytic			+	Well 10 years (case lost).
F	45	Buccal mu.	Reticulum			+	Died 17 years 3 mos. No recurrence.
M	27	Nasopharynx	?	+			Well 10 years (case lost).
M	45	Rectum	Giant follicle		+		Well 14 years 1 mo.
M	64	Stomach	Reticulum		+		Well 15 years.
M	20	Stomach	Reticulum	+			Well 17 years.
M	40	Stomach	?			+	Well 10 years 9 mos. (case lost).

R=Radiotherapy alone. S=Surgery alone. RS=Both surgery and radiation.

TABLE V

25 Patients with Lymphosarcoma known to have lived more than 10 years

		Living and well over (years)								Died other causes over (years)			Died disease over (years)
		10	11	12	13	14	15	16	17	13	16	17	10
Lymph Nodes													
Cervical	5	.....	1	2	1	.....	.....	.....	.....	.....	1	.....	.....
Axillary	3	.....	.....	1	1	.....	.....	1	.....	.....	.....	.....	.....
Inguinal	3	1	.....	.....	.....	.....	.....	.....	1	.....	.....	.....	1
Retroperit.	1	.....	.....	.....	.....	.....	.....	1	.....	.....	.....	.....	.....
Generalized	1	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	1
Mouth and Nasopharynx													
Cheek	2	1	.....	.....	.....	.....	.....	.....	.....	.....	.....	1	.....
Tonsil	2	.....	.....	.....	.....	.....	.....	1	.....	1	.....	.....	.....
Nasopharynx	2	1	.....	1	.....	.....	.....	.....	.....	.....	.....	.....	.....
Gastrointestinal													
Stomach	4	1	.....	1	.....	.....	1	.....	1	.....	.....	.....	.....
Ileum	1	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....
Rectum	1	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....
TOTAL	25	4	1	5	2	2	1	3	2	1	1	1	2

patients are cured. It seems significant, however, that our records include only two patients living over ten years after treatment who subsequently died of the disease. It is at least legitimate to hope that the others may have actually been cured. It can be seen from Table IV that the types of treatment used include surgery alone as well as radiotherapy and a combination of the two. This fact also suggests that the lesions treated may have been focal and not simply manifestations of generalized disease.

Table VI deals with 212 patients suffering from Hodgkin's disease observed at the Presbyterian Hospital during the years 1915-1943 inclusive; 180 of them were treated by radiotherapy and 32 had no attempts at curative therapy. All of the untreated cases are dead; 31 of them during the first 3 years after observation and the other during the 9th year. 132 of the 180 treated patients or 73.3 per cent died or were last seen alive less than five years after onset. The other 48 have lived past five years. Half of them are known to be dead but some have lived a long time before they died with Hodgkin's disease — 7 of them over 11 years and one of the 7 for 19 years. The other 24 at last reports were alive, some of them with disease still evident but others without. There are 10 such patients who have been symptom free following onset for the following periods of years: 8 1/12, 8 10/12, 9, 9 6/12, 10 3/12, 10 6/12, 11 2/12, 16, 16 3/12, 17 6/12. Four other patients were alive with disease still persisting one in the 9th, one in

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TABLE VI

HODGKIN'S DISEASE — 212 CASES

Presbyterian Hospital, New York, 1915 — 1943
Duration of Disease from Onset

Treated by Radiotherapy — 180 Cases

	0-12	13-24	25-36	37-48	49-60	61-72	73-84	85-96	97-108	109-120	121+
Died	22	37	16	16	11	6	3	3	2	3	7†
Alive	14	7	3	5	1	4	2	4	3b	3a	8*
	36	44	19	21	12	10	5	7	5	6	15

No Treatment or Palliation — 32 Cases

Died	15	3	3	.....	.....	.....	.....	.....	1	.....	.....
Alive	7	2	1	.....	.....	.....	.....	.....	.....	.....	.....
	22	5	4	.....	.....	.....	.....	.....	1	.....	.....

*6 cases apparently symptom-free 17 1/2, 16 3/12, 16, 11 2/12, 10 1/2, 10 3/12.
2 cases alive with disease 10 10/12, 10 4/12.
†Died 19, 18, 15 2/12, 13, 12, 12, 11, all with disease.
a—2 cases symptom-free at 9 6/12 and 9 years.
b—2 cases symptom-free at 8 10/12 and 8 1/12 years.

HYPOGLYCEMIA*

Prevention, In the New Diabetic, With Early Stabilization On Low Fat Diet, With Resultant Low Insulin Dosage

LOUIS E. BURNS, M.D.

The Author. *Louis E. Burns, M.D., of Newport, R. I. Physician-in-chief, Newport Hospital; Past President, Newport County Medical Society.*

"Hypoglycemias, caused by overdoses of insulin, entail in the diabetic patient excessive degrees of hyperglycemia and glycosuria. Recurrence of this sequel over considerable periods of time progressively increases the instability of the patient and aggravates the disease."

MICHAEL SOMOGYI[1]

ADHERING to the underlying principles of the above quotation for successful treatment of the new diabetic, many basic physiological facts have to be applied.

The basic principles are: the role of the liver; avoidance of hypoglycemia or insulin reactions; importance of daily fractional urinalysis; value of blood sugar examination and diet.

By successful treatment of the diabetic, I mean sufficient food to prevent hunger and dietary invalidism, maintenance of normal body weight and strength, keeping the urine sugar free, continuation of the daily occupation and if insulin is required, keeping the dosage low enough to preclude any hypoglycemic reactions.

I think from our experiences and methods of treatment so far, it is safe to say that the first physician that treats the new diabetic is the master of his or her destiny. I say this because of what I have seen over the past few years. There are too many early cases made unmanageable because of over-insulination or neglect. I think that by a clearer understanding of some of the underlying physiology of carbohydrate metabolism in the liver, the new diabetic can be kept a useful, self-supporting citizen and will never become so unfortunate as to have to be rehabilitated.

After the discovery and use of insulin, it didn't take many years of this therapy for us to realize that insulin alone did not do all that was expected of it in diabetes, so the natural conclusion was that some other organ must be involved. Therefore, the unitarian theory that the pancreas alone was the sole organ involved in diabetes mellitus was discarded.

In 1923, Mann and Magath[2] proved beyond any doubt the role of the liver in diabetes. They found that dogs that were depancreatized had as rapid a fall in blood sugar after the removal of their liver as did the normal. They also showed that these dogs died identically within a few hours in convulsions and both had low blood sugars (hypoglycemia). This paper gives a background to the role of the liver in maintenance of blood sugar in human beings. In 1850, Claude Bernard showed by his work, that glycogen in the liver was responsible for maintenance of normal blood sugar, thereby proving that the liver is the storehouse for glycogen.

The glycogen[3] content of the normal liver varies greatly, being greatest after the intake of a heavy carbohydrate meal, less when a high fat meal is eaten, least when all food is witheld.

The liver[4] is the predominant organ in the regulation of blood sugar concentration, since it is the only organ that possesses a store of glycogen that can be rapidly converted into glucose and is capable of forming glucose from non-carbohydrate sources, such as proteins and fats, in quantities sufficient for the bodily needs.

The normal glycogen[5] content of the liver ranges from 100 to 400 grams, depending upon the size of the individual.

Liver in Diabetes

An autopsy[6] analysis of the fat in the livers of diabetic and non-diabetic patients by Dr. Halliday, at the Deaconess Hospital, showed the total fatty acid to range from 2.2 to 4.3 per cent net weight in the non-diabetic and from 4.1 to 10.8 per cent in the diabetic cases. These figures are very significant for our work with the new diabetic on low fat diet and our method of treatment.

Fat deposit in the liver is influenced by: diet, starvation; insulin deficiency and lack of the lipotropic substances, i. e. methionine, choline, inositol.

Hypoglycemia Reactions on the Liver of Diabetics and Why they are to be Avoided

It has been shown in the course of a four hour, 100 gram glucose tolerance test on normal indi-

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viduals that hypoglycemia occurs one to two hours after the hyperglycemic peak has been reached, only to be followed by a secondary rise above the original fasting blood sugar level, then to hypoglycemia again. This rise and fall continues to occur until a balance is attained, hence the phrase "Hypoglycemia begets hyperglycemia".[1] The same thing holds true in the diabetic, but to a greater degree (because the glycogenolysis is greater). In the normal individual, after meals or during fasting or in the post cibal period, the blood sugar is kept within normal limits by the endocrine system.[7]

Hypoglycemia, produced by an overdose of insulin in the diabetic[1], causes a great disturbance between glycogen storage, i. e. glycogenesis and glycogen breakdown in the liver, which is glycogenolysis, because in the diabetic this endocrine balancing system is faulty. We must remember that the liver is the storehouse for glycogen, and glycogen release is what overcomes the low blood sugar level and restores it to the normal.

When the insulin action is waning, the liver glycogen breakdown[1] i. e. glycogenolysis goes on at a terrific rate, with the result that the following hyperglycemia does not slow down or stop at the normal level, due to the endocrine system being out of balance, but the blood sugar continues up beyond the kidney threshold level and produces glycosuria.

This insulin produced shock, if continued, produces instability in the diabetic and the disease is made worse.

Overdosage of insulin not only produces a low blood sugar, followed by high blood sugar and sugar in the urine, but goes further and helps deplete the glycogen reserve in the liver. As we know, an unstable diabetic has a low glycogen reserve and shows ketosis and ketonuria[17] on the slightest provocation, whether it be from exercise, fever[16], excessive insulin dosage[1], starvation or very low carbohydrate intake. If the insulin induced shocks are produced often enough, deep enough and long enough, the following hyperglycemia and glycosuria is accompanied by ketonuria.

How is this insulin induced hyperglycemia and ketosis avoided? We know that the chief source of ketone body formation is in the liver[15], so ketosis following a prolonged severe insulin shock always accompanies the rapid rate of hepatic glycogenolysis[17]. The only way to relieve this reaction is to administer glucose in good quantities to bring the blood sugar up to the hyperglycemic level, for it has been shown that hyperglycemia stops glycogenolysis. There is another way to prevent hypoglycemia—that is to examine every sample of urine passed for sugar. For instance, a morning sample before breakfast contains a 2% glycosuria and the patient's insulin dosage is 30 units (PZ 20 and CZ 10). As the day goes on and the urine shows a decreasing glycosuria to ¼% between one and three P. M., we know from experience that before long the urine is going to be negative, because the blood sugar will be below the kidney threshold level and may continue down into hypoglycemia, so the treatment right here should be orange juice in good quantities. A good rule to follow in the face of a rapidly decreasing glycosuria any time during the day, is to always give orange juice to prevent hypoglycemia. If the glycosuria has decreased to negative in the evening from a morning 1% to 2%, give orange juice immediately, and at bedtime 15 grams of carbohydrate with 5 to 10 grams of protein should be taken for the lasting effect to forestall the night insulin shock. For this reason, night feedings are always prescribed when the long lasting protamine zinc is given in the morning.

Hypoglycemia

I cannot speak loudly enough, strongly enough and often enough about the avoidance of hypoglycemia in diabetic patients. It brings on a chain of symptoms that may leave the patient a chronic invalid for the rest of his natural life. It is a cardinal sin and must be avoided for the successful treatment of the new diabetic. The results may be ruinous to the human body and the worst damage is found in the brain. A patient's father once said, "Those insulin reactions are far worse than the diabetes." Truer words were never spoken.

Dr. Himwich[8] in his review of hypoglycemia reactions, said, "Irrespective of the cause of hypoglycemia, the symptoms are the same and involve the brain chiefly. Brain metabolism is depressed and cerebral function cannot be maintained." From the medicolegal[10] standpoint, the brain shows the most marked changes, plus a marked increase in liver glycogen.

It has recently been shown that Labile Diabetics[9] exhibited symptoms of epilepsy irrespective of their insulin dosage or diet. Control of the diabetic state was brought about only by the use of anticonvulsants.

Dr. Joslin[6] summed up hypoglycemia by saying, "Diabetic coma may kill people, but frequent hypoglycemic reactions will ruin them."

The medical profession for years has treated diabetes by[11] one sign alone, glycosuria, without uniform or satisfactory results. We still do urine examinations, but on every sample passed. Every sample of urine voided is tested and recorded as to time, volume and percentage of glycosuria present regardless of whether day or night.

Blood Sugar

The one morning fasting blood sugar level examination alone is useless and dangerous, for many physicians base their insulin dosage for the day upon this single examination. Frequent blood sugar

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tests at different times in a day give us much more information, and the progress of diet and insulin is followed more carefully and scientifically in the body. It is better to know what the blood sugar is doing before and two or three hours after a meal, or at bedtime, until the disease is stabilized.

Now we have two tests to help us, the urine that shows us the time and amount of glycosuria, and the blood sugar that tells the level at that time.

By knowing the result of these two tests at various times during the day, especially if the patient is on insulin therapy, hypoglycemic reactions can easily be averted.

When a new case of glycosuria is seen in our clinic at the Newport Hospital, the first thing we do is to exclude all other conditions that may produce sugar in the urine—then the four hour 100 gram glucose tolerance test is done. If a definite curve of diabetes mellitus is found, our attention is then turned to treatment. When we think of treatment, the organs we talk about are the liver and its glycogen content, the pancreas and its insulin production, for, as we all know, insulin and glycogen are dependent upon each other.

We lean toward the liver, for as our treatment progresses, the diet is nearly that of the cirrhotic dietary treatment. In a new and untreated diabetic, there is an uneven distribution of glycogen between the extra cellular fluid, cytoplasm and nucleus of the liver cell. Most of the glycogen is found in the nucleus of the cell, but after therapy, it moves to the cytoplasm and a more even and normal distribution takes place. The glycogen reserve in the liver of a new diabetic is greatly diminished, because it is not storing glycogen well, for if it were, there would not be any hyperglycemia and glycosuria. Therefore, the liver is in a state of glycogenolysis, that is, putting out sugar. To achieve successful results in treatment, we have to set about and reverse this process of glycogenolysis and put the liver in a condition to store glycogen. This process is called glycogenesis.

The diets I have used are moderate to high carbohydrate, liberal protein and low fat. The reasons for this are obvious if one thinks in the terms of impaired liver function in a metabolic disorder like diabetes mellitus and that, over the course of years of treatment, nutritional disturbances occur.

In the treatment of metabolic diesases today, more and more attention is being focused on the liver. A tremendous amount of research has been done in cirrhosis of the liver. It has been found that this organ became infiltrated with fat, and its function was decreased in many instances to a point of failure.

In many untreated, poorly managed and uncontrolled cases of diabetes, the liver has much fatty infiltration, with greatly impaired carbohydrate metabolism, especially glycogen storage. There seems to be some mechanico-chemical inhibition of insulin action. Today, with our long lasting insulin and low fat diet, this condition is not as evident as when insulin of short duration was used.

The fat in our diet amounts to between 50 to 65 grams, which is enough to supply the daily fatty acids for the body. When the excess of fat in the liver is decreased to normal, glycogen storage improves.

The protein in the diet of the cirrhotic is generally two or more grams per kilogram of the body weight per day. The protein in our diets runs well above the conventional one gram per kilo for adult diabetics. We give from 1.5 to nearly 2 grams of protein per kilogram of body weight.

Children are fed the usual 2 to 3 grams of protein per kilogram of body weight, which is so necessary for body growth. The protein of our diet is varied and high enough to supply all the amino acids and prevent hypoprotenemia.

In my diets, insistence on the inclusion of good quantities of skimmed milk and cottage cheese is made[12]. The casein[13] from cottage cheese supplies lipotropic substances—methionine[14] which, with transmethylation, leads to the formation of choline, necessary for the prevention of the accumulation of fat in the liver.

The carbohydrate content of the liver diet runs between 250 and 400 grams per day. I use, in our diet, from 200 to 400 grams daily of carbohydrate. Our patients seem to do better when they get from 200 grams to 300 grams of carbohydrate per day, and as the liver begins to store glycogen, the need for insulin decreases.

My carbohydrate allowance is very liberal after stabilization. I allow sugar in tea and coffee, hard candies, soft drinks—especially to people doing heavy manual labor. Boys and girls of dancing age should take soft drinks during the evening to forestall any hypoglycemia from the strenuous exertion. We insist on this and to date have not had any hypoglycemic reactions.

One patient who was in precoma, after being treated for 10 months on our treatment, was stabilized on 6 units of protamine zinc insulin daily. He was given a diet of 450 grams of carbohydrate (which is nearly one pound), 133 grams of protein and 66 grams of fat — total calories 2726 — and showed only an occasional quarter per cent glycosuria during the week. This man's liver, in my opinion, was brought back to the nearly normal for carbohydrate metabolism.

Discussion

I am purposely omitting the word "mild" from my diabetic vocabulary. I do not think clinically

there is such a thing. I admit that all cases start mildly, but when they get to the point where the disease can be diagnosed clinically, they then are beyond the so called mild stage and should be treated as diabetics, without qualification. All diabetics that have glycosuria have the same trouble in the liver, that is difficulty in storing glycogen. Insulin helps to restore this mechanism to normal. I don't mean to fill up diabetics on insulin in hit or miss fashion, and let them go on using their own judgment, but they should be under good supervision, examined frequently and kept on small doses of insulin to favor glycogen storage and build a good glycogen reserve in the liver.

As I have seen them over a period of years, the tendency of the cases, which have been brought down to the point where they could get along without insulin, is that they feel well, in fact too well, and then comes the neglect. This is first shown in non-attendance at clinic, next nibbling just a bit more food. They think they can get away with it, because they didn't have glycosuria, and it isn't long before they go "all out", breaking the diet completely. These individuals begin to feel tired and sluggish, sugar appears in the urine and in good quantities, and they are completely out of control again. If I try to get these people back on insulin and a good regime again, they offer all sorts of excuses and finally they say, "You said I was a mild diabetic. Why don't you treat me as such, with diet alone? I don't want that needle again."

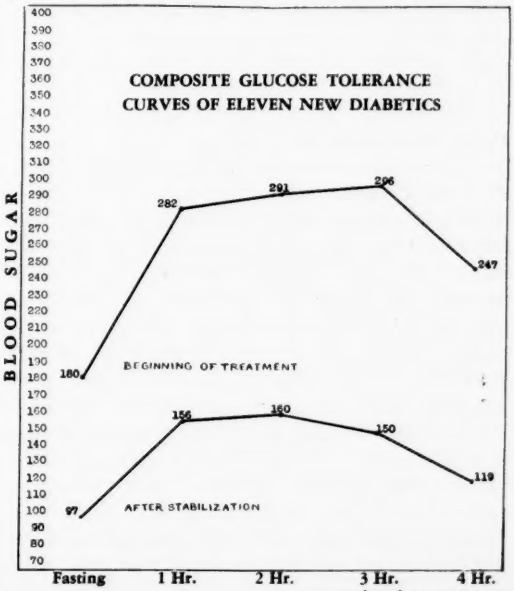
These are childish arguments, but whose fault is it when they get out of control again? Ours, because when we had them under good control and aglycosuric on small doses of insulin, that is 5-6-7 or 8 units of insulin daily, we should have kept them there. The main thing in the treatment of these so-called "mild" diabetics is never to let them lose sight of the fact that they are still diabetics, but under excellent control.

I have one case in mind, a woman, that was brought down from 30 units of insulin daily to zero over a period of 12 months. This woman was in her fifties. After six months on no insulin and theoretically under good control, as shown by aglycosuria, she became more and more careless about her diet as to restricted foods. Within another few months, her blood sugar levels were high again, both fasting and after eating, glycosuria appeared in good quantities and she was completely out of control. For the next 12 months she refused insulin and at this time complained of difficulty in vision. Careful eye examination disclosed a far advanced diabetic retinitis (practically blind), whereas at the time of discovery of her diabetes two years previous, the retina was perfectly normal. This diabetic degenerative change, which took place within two years, normally occurs after fifteen years of the disease. When these people are brought down to a low daily insulin dosage, doses that were heretofore considered to be too small to be of any value, the taking of these small doses made the patient realize that he was still a diabetic, and had a psychological value in making him follow the treatment faithfully. Therefore, he enjoyed continued good health, for he was controlled. Most diabetics, when cut loose from restriction, go adrift and become lost forever. They are the potential candidates for the degenerative changes of diabetes, namely—retinitis, arteriosclerosis, nephritis, hypertension, coronary disease and polyneuritis.

It is difficult to understand why a diabetic's blood sugar should not be kept within the same limits which we establish for normal people. It is in the same category as any other abnormality of blood chemistry, such as the NPN, urea and uric acid.

We know what the normal physiological limits are, and it seems only logical to attempt to restore all sorts of patients to within these physiological bounds, if there are means available to do so. It is well-known that the patient, who becomes sugar free, manifests an improvement in carbohydrate tolerance and the need for insulin is lessened. I have tried to show how the new diabetic can accomplish this result and remain sugar free under good careful management.

By keeping the blood sugar and cholesterol of diabetics within the normal range, after a period of treatment, by means of low fat intake and the use of lipotropic forming substances in the diet, together with low dosage of insulin, we can keep the liver in a continuous state of storing glycogen. By this procedure, we may be on the right road to prevent the diabetic from developing degenerative



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diseases earlier in life than the non-diabetic. Time alone will tell.

These cases are not hand picked for statistics, but represent all of the newly diagnosed cases that came into our clinic at the Newport Hospital and followed the treatment mapped out for them. They range in age from ten years to sixty odd. They represent 100% success on our treatment. In the past three years, I have had 33 new cases. Out of these 33 newly diagnosed cases, only 11 stayed. It shows that about one-third of the number of diabetics are willing to be guided and helped. Of the remaining two-thirds, only God knows what is happening and will happen to them.

Statistics show that about 60% of diabetics die of arteriosclerosis. In my own experience, about two-thirds of the number of diabetics will not carefully follow the treatment laid out for them. These two percentages are fairly similar, and lead me to wonder whether one might not draw the conclusion that it is the diabetic who neglects his treatment that is largely responsible for the high ratio of arteriosclerosis. We know that diabetics are more liable to develop arteriosclerosis much earlier than the non-diabetic. Why? We don't know, but this fact alone points out the necessity of an intensive educational campaign in the case of the new diabetic. Future statistics on the prevalence of arteriosclerosis in diabetics adhering to the diet and treatment oulined herein, should prove most interesting.

Summary

In our cases, it may be seen that the commonly accepted insulin dosage, which inevitably causes hypoglycemia, could be substantially reduced, since tolerance for carbohydrates was distinctly increased. In the majority of the new diabetic cases that came to us initially, we gave from the outset only moderate doses of insulin—just sufficient to enable the patient to metabolize adequate amounts of carbohydrate. Hypoglycemia was carefully precluded by intermediary feedings, if necessary. As the glycosuria showed a noticeable diminution, the insulin dosage was correspondingly decreased in moderate steps and the carbohydrate and protein content of the diet was increased. The end result was that in several cases, the patient remained aglycosuric without the use of insulin. In other instances, the high insulin dosage started with, has gradually been reduced to a lower level, and as treatment is continued, will be further decreased.

These eleven new cases of diabetes represent 100% success on our treatment. Insulin dosage, at beginning of treatment ranged from 0 to 60 units per day—average 26.5 per day. Insulin dosage at end of treatment, or after stabilization, ranged from 0 to 14 units per day—average 6.5 units per day.

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The RHODE ISLAND MEDICAL JOURNAL

Owned and Published Monthly by the Rhode Island Medical Society,
106 Francis Street, Providence, Rhode Island

THE NEW MEDICAL BUREAU

THE ACTION of the Providence Medical Association in establishing in the Rhode Island Medical Society building a 24 hour telephone secretarial exchange for physicians represents a potential community service that will exceed the direct benefit that may accrue to the individual physician subscribers.

The enterprise is a major one for the Providence physicians to undertake, but the enthusiastic response that has met the appeal for members to join the Bureau indicates that the venture will be a successful one from its very start. Primarily a service whereby telephone calls are covered around the clock, the Medical Bureau proposed by the Providence Medical Association will render a wider service than the commercial exchanges that have operated in the area can offer.

A classified list of physicians who are members of the Association will be made available to the Medical Bureau for the convenience of the public. Any active member in good standing may be listed, and on inquiry a caller will be given the names of three physicians from this list. Every effort will be made to refer the caller to his personal family physician before the information is provided, and when names are given a rotating system will be followed to guarantee that the names of physicians are given an equal number of times.

Aside from the usual inquiries about physicians and medical facilities, the Bureau anticipates an Emergency Call service whereby individuals who do not have a family physician and who need immediate medical attention may secure help. A list of members of the Association willing to answer such emergency calls will be prepared, and utilized on a geographical basis throughout the city to expedite the service and eliminate unnecessary travel by the physician.

As a cooperative venture the Medical Bureau warrants the active support of every member of the Providence Medical Association. The complaint is often heard that strangers in our city, and even residents of long standing, are unable to secure reliable information at one source about reputable physicians and medical facilities. This situation will undoubtedly be remedied by the new Bureau. With direct lines anticipated to the large hospitals, to the city health department and the police department and with experienced operators on day and night duty, trained to handle the calls with judgment and tact, the Bureau represents an outstanding line of communication between the public and the medical profession.

THE NARCOTIC REGULATION

We have just straightened out the business of our Narcotic license, so naturally we are feeling hot-under-the-collar. Our use of narcotics should be, and practically always is a boon to humanity. Probably for purpose of regulation it is advisable that we should be licensed. When we make our annual statement that we have a couple dozen morphine tablets on hand it is a great deal of a nuisance that we and Mr. Johnston, the Notary Public, have to interrupt our useful activities to have the statement notarized. It is hard for us to believe that it serves any useful purpose.

Why not have our prescription notarized every time we write for fifty doses of morphine for a cancer patient in the last stages? There is a great deal more chance for cheating and checkups should be much more requisite in this last transaction. But, the most ridiculous thing of all, the child of some small idle bureaucrat mind, is the certification of our dollar check for a license. This cannot serve any useful purpose. There is nothing delivered to us before our check goes through the regular channels and is proven good. If we don't send in a good check, our license can be taken away, we can be fined or thrust into prison.

Think of the wasted hours of the men presenting the check and the busy bank clerks going through all this folderol seven hundred times, repeated each year. But, this does give us an insight into what we will all have to endure if medicine is made bureaucratic.

DEMOCRACY AT WORK

We certainly hope that the newspapers who, in the past, have charged medicine with being "organized" and that the A. M. A. is dictatorial, sent reporters to cover the recent House of Delegates meeting at Atlantic City on the occasion of the A. M. A. Convention.

All the meetings were open to the press. There was no "Executive Session."

The Board of Trustees of the A. M. A. must carry out the mandates of the House of Delegates. The House of Delegates is composed of representatives—one for each thousand physicians from the various states. The most significant fact about the operation of the House at the last meeting was the fact that of 170 delegates accredited from every state including Alaska, Porto Rico and Hawaii, less than six were absent whenever roll was called. These men not only attended meetings of the House diligently and attentively but took active and sometimes vociferous part in the proceedings. On two occasions it was necessary to have a standing vote and the ayes and the nays had to be counted, so close was the balloting on important questions.

For those who scoff and say the A. M. A. is not representative of medicine, the answer should be apparent that the A. M. A. represents those who really want to be represented. In a democracy if the delegate doesn't represent his constituents he needs to be replaced and should be.

The second and equally important fact that was obvious at the Convention was that any Delegate could seize a microphone at anytime and talk on the subject before the House and, by full and frank discussion, all points of view could be aired.

This last session was truly an example of Democracy at Work.

SCANDAL WITH HAPPY OUTCOME

After a short courtship they were united in February 1826, the synchronous result of the union being the Boston Medical and Surgical Journal, born on the nineteenth day of the same month.

Editorial in *New England Journal of Medicine*

Almost in every kingdom the most ancient families have been at first princes' bastards; their worthiest captains, best wits, greatest scholars, bravest spirits in all our annals, have been base (born).

Anatomy of Melancholy
Burton via Bartlett.

In a recent editorial our staid Boston matron has made some astounding revelations regarding the family history. In the same number they have flaunted for the first time a coat of arms. Our knowledge of heraldry is practically nil. We cannot say whether any of the insignia on this escutcheon shows a bar sinister. Probably not for it seems to be well-recognized that a shotgun marriage makes an honest woman.

We are sure that Shakespeare, in speeches that we cannot now turn to, extols the virtues and brilliances that have got their genesis outside prosaic marriage beds. The very excellences of our admired sister publication may now be seen to support that theory. Once again we express our admiration and respect.

MEDICAL EXAMINER LAW

In 1939 the General Assembly abolished the office of coroner and substituted a medical examiner system. Now, ten years later, the Assembly has revised the medical examiner system, incorporating most of the recommendations of a joint committee of the Rhode Island Bar Association and the Rhode Island Medical Society. The new system under a chief medical examiner for the State was effective on July 1, and on page 456 the list of deputy examiners named for the various counties is published.

continued on next page

It is interesting to note, however, that in what was supposed to be an improved system the General Assembly failed to make adequate financial provisions to carry on the program it spelled out in the statute it adopted. Provision was made for the office of chief medical examiner and the restrictions placed upon the person filling that office are hardly duplicated in any other assignment in State service. For example, the chief examiner "shall devote his full time to the job, he shall not engage in other gainful employment, he can be removed from office for cause, he must be qualified in pathology and have had at least a year of medico-legal training. And for such a highly-trained specialist a top salary of $7,500 is set. And since the General Assembly appropriated only $9,000 for all the improvements outlined in the new statute, just how a central office and a post mortem laboratory within the city of Providence can be financed on the funds available is a question unanswered at this time. For the present, at least, the chief medical examiner will have to operate his new position from what has been his own private office.

While the Assembly was not liberal in providing funds for carrying out the new program at the level proposed by the joint committee of the bar and medical societies, it was most generous on another issue where the joint committee advocated restraint.

Conscious of the difficulty that could, and did arise, in securing the services of a medical examiner promptly when a person died suddenly on a public highway from apparent natural cause, the joint committee recommended that the removal of such body to an appropriate place may be ordered *by any superior police officer* as well as by others *now having* such authority. But the Assembly wasn't satisfied to have the superior police officer act in such cases. It provided that "any member of the family of the deceased, relative, next of kin, neighbor, acquaintance or any superior police officer" can move the body. That just about makes everyone eligible to determine when death on the highway is apparently from natural causes.

LYMPHOSARCOMA AND HODGKIN'S DISEASE

concluded from page 439

the 10th, and 2 in the 11th year following radiotherapy. Is it possible to suppose that any of the long survival symptom-free patients are cures? It would be hazardous to affirm it when it is noted that so many have survived equally long times with the disease still persisting. What can be stated with assurance is that treatment by radiotherapy has certainly lengthened these patients' lives for not a single untreated case lived as long as 10 years.

In summary therefor it can be affirmed that while both lymphosarcoma and Hodgkin's disease are processes which are invariably fatal if untreated, a small number of cases seem to have a focal origin and progress slowly. Prolongation of life has unquestionably been achieved both by radiotherapy and surgery in the case of lymphosarcoma and by radiotherapy for Hodgkin's disease. Presumptive cures have resulted in cases of lymphosarcoma lasting from 10 to over 17 years, although evidence is still lacking as to whether or not these are absolute cures. For Hodgkin's disease it is not possible to suggest that the long survival symptom-free cases are presumptive cures because of the number of equally long survival patients with persisting disease.

For both lymphosarcoma and Hodgkin's disease it is possible that there are two clinical varieties which have the same histopathological picture but run a very different course. In one the disease is widespread when first observed or shortly thereafter and the patients die usually in 5 years or less from the time of onset. Unfortunately this is the commoner experience and so far treatment has failed to cure any of the victims. A much smaller group exhibits a focal origin which sometimes remains restricted to the primary site and adjacent areas for quite a period of time. The examples of long survival cited above in the great majority of instances have belonged to this more favorable but much smaller group.

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THE AMERICAN MEDICAL ASSOCIATION MEETING

Report of the Delegate from Rhode Island, Charles L. Farrell, M.D., on the meeting of the House of Delegates of the AMA, at Atlantic City, June 6-9, 1949

THIS was the second largest Convention in the history of the American Medical Association. Of the total registration 13,221 were physicians, 15,000 were visitors. 58 Doctors registered from the State of Rhode Island. There were 300 scientific papers, 212 scientific exhibits, 341 technical exhibits including color television.

Dr. Ernest E. Irons was elected President, Dr. Elmer L. Henderson, President-Elect, Dr. Louis Bauer was made Chairman of the Board of Trustees. Dr. F. J. L. Blasingame of Wharton, Texas, was elected to the Board of Directors to fill the unexpired term of Dr. Henderson. Dr. George Lull was re-elected Secretary.

The highlights of the activities of the Session were as follows:

1. Dr. Fishbein was supported for his past contribution to the profession but his activities were now limited to being Editor of the Medical Journal and he is not to be the spokesman for the profession.
2. They re-established the Committee on General Practice and re-affirmed the need for general practice sections in hospitals.
3. The A. M. C. P. was separated from the American Medical Association and established as a separate trade organization. Twenty principles for lay sponsored health plans were adopted, a copy of which is hereby enclosed.
4. The resolution for displaced persons introduced by resolutions from Rhode Island and New York were referred to the Board of Trustees for appropriate action.
5. The House defeated the resolution to stop the general practitioner award in spite of the petition from the Connecticut Medical Society to do so.
6. They revised the principle of medical ethics: copies of which have been published in the July 2nd issue of the AMERICAN MEDICAL ASSOCIATION JOURNAL.
7. They opposed the inclusion of doctors in Social Security legislation.
8. The attempt to provide contract coverage of veterans for medical and hospital care provoked much debate. This resolution was introduced by Tennessee and, although supported to the Reference Committee, it was defeated on the floor of the House by a vote of 61 to 59. The subject was reopened the next day and after much debate was tabled by a vote of 74 to 69.
9. The subject of insurance fees was discussed at length but no action was taken. Dr. Dickinson reported that there was no national agency to which the dispute could be referred and he is sending a direct report on this matter to the Secretaries of the District Societies.
10. The House approved the report which recommended that each State and District Society form a Committee on Hospital and Professional Relations.
    It was the feeling of the Reference Committee that doctors were properly interested in the finances of the hospital, that every man on the staff of every hospital had a vital interest in the operation of the hospital, and that by the democratic process should make his voice and opinions heard thru the Executive Committee. It strongly recommended that the local societies, thru their Committee on Hospital and Professional Relations, investigate and adjudicate complaints regarding the conduct of hospitals and hospital staffs. The Committee also affirmed its position that hospital plans should pay only for hospital services, that medical plans should pay the bills for services of physicians, and they did much to clarify the thinking on the proper fields of coverage for insurance programs covering medical and hospital services.
11. The following members from Rhode Island were made Associate Fellows:
    Dr. C. O. Cooke of Providence
    Dr. S. A. Kenney of Valley Falls
    Dr. B. F. Tefft of West Warwick
    Dr. C. S. Turner of Providence
    Dr. J. L. Wheaton of Pawtucket
12. Dr. McVay of the Council of Medical Service reported that there are 30 million people now covered by Blue Cross and 10 million people by Blue Shield. The total people having hospital coverage was well over 55 million. 37 million had coverage for surgical or medical service.

continued on page 452

THE A.M.A. MEETING

concluded from page 450

Twenty Principles Listed

The twenty suggested principles for lay sponsored voluntary health plans follow:

1. The plan shall be non-profit, pay no dividends to beneficiaries or others; all surplus earnings shall be devoted either to improving services, to making compensation of physicians and other staff members more adequate for their responsibilities and services, to purchasing facilities and equipment, to increasing the scope of benefits or to building annual reserve funds. All income to the plan shall be devoted to services for beneficiaries.

2. The plan shall comply with the principles of medical ethics of the American Medical Association, which provide that it is unprofessional for a physician to dispose of his professional attainments or services to any lay body, organization, group or individual, by whatever name called, or however organized, under terms or conditions which permit a direct profit from the fees, salary or compensation received to accrue to the lay body or individual employing him.

Capital Stock Barred

3. If incorporated, the plan shall be adequately financed and organized without capital stock.

4. The plan shall be operated under an autonomous administration or trust, with segregated funds, and shall be devoted exclusively to the provision of helath service.

5. Promotion, sales, organization and administrative expense of the plan shall be kept at a minimum as judged by the accrediting body.

6. The quality of medical service shall be maintained at the highest possible level. All participating physicians shall be doctors of medicine duly licensed to practice medicine in any state in which the plan operates. Each physician engaged in the practice of a specialty shall be required to have adequate qualifications for that specialty. The personnel and facilities of the plan shall be adequate to insure a high quality of medical care.

Provison for Service

7. The plan shall provide all services as set forth in the agreement with the beneficiary. state in the opinion of the medical staff, a professional service set forth is not available because of an emergency or because of the need for highly technical procedure, or for any other reason, then such service shall be otherwise provided by the plan.

8. The plan in its agreement entered into with the beneficiary and which shall be distributed to each beneficiary, shall state clearly the services and benefits to be provided and the conditions under which they will be provided. All exclusions, limitations, waiting period and deductible provision shall be clearly stated in the agreement with the beneficiary and in promotional and descriptive literature.

9. The plan shall, in its agreement with the beneficiary, state clearly the amount of dues or subscription to be paid. The amount of dues or subscription shall be adequate to provide for the benefits and services offered and to insure proper financing of the risks involved.

10. No promotional material shall invite attention to the professional skill, qualifications or attainments of the physicians participating in the plan.

Compensation to Doctors

11. Participating physicians may be compensated in any manner not contrary to the principles of medical ethics of the American Medical Association relating to contract practices.

12. Any duly licensed physician in the community who wishes to participate in the plan. who meets its professional and personnel standards, and who agrees to abide by its terms and the requirements of its beneficiaries, shall be admitted to the plan.

13. The names of all participating physicians of the plan shall be made available to the prospective beneficiary. The beneficiary shall, within reasonable geographic and professional limitations, have free choice among participating physicians.

14. There shall be no interference by the governing body with the medical staff in the practice of medicine. The traditional and confidential relationship of the physician and patient shall be preserved.

15. Adequate provisions shall be made for effective participation of the medical staff in the deliberations of the governing body. It is recommended that the membership of the governing body include representatives of the medical profession.

16. All services rendered by the participating physician, not included in the beneficiary's contract, shall be payable by the beneficiary to the participating physician on a fee for service basis.

17. The method of operation of any hospital owned or under contract to the plan shall be in accordance with sound public policy.

18. The plan shall provide for like rates, benefits, terms and conditions for all persons in the same class.

19. Investment of reserve funds shall be made only in securities deemed prudent for such purposes.

20. Any plan desiring approval under these principles shall agree to such periodic reviews and to abide by such regulations as may be deemed necessary by an appropriate accrediting body of the American Medical Association in consultation with representatives of the sponsors of the plan.

CHARLES L. FARRELL, *Delegate*

KENT COUNTY MEDICAL SOCIETY

The May meeting of the Kent County Medical Society was held on Tuesday, May 17, 1949, at the home of Dr. Hardy.

Prior to the regular meeting Dr. Rocco Abbate gave a résumé of the controversy between the R. I. Medical Society and the Blue Cross over the prepaid surgical insurance.

The meeting was called to order by the president and a short business session was held. Dr. Joseph C. Kent was elected vice-president to fill the unexpired term of Dr. Francis D. Lamb, who is on a leave of absence from the Society. Dr. Jean M. Maynard of 40 Curson Street, West Warwick was elected secretary to replace Dr. Kent.

Drs. Taggart, Mack and Hudson were appointed by president Dr. Hardy as a committee to arrange for the annual clambake to be held in June.

Following the business meeting two reels of moving pictures were shown. These pictures, produced by Parke, Davis & Company, showed the use of Oxycel in caeserian section, and Thrombin as a hemostatic in a case of evulsion of the entire scalp which was skin grafted.

The meeting adjourned at 11:15 p.m.

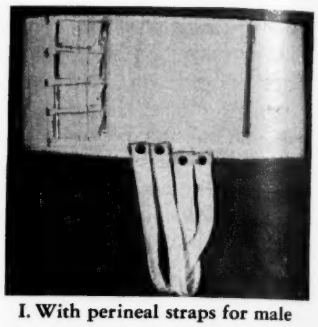
Respectfully submitted,

JOSEPH KENT, M.D., *Secretary*

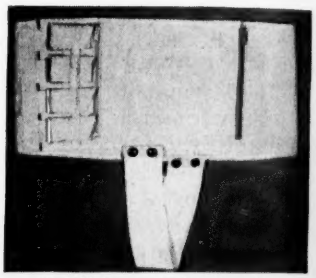
AN IMPROVED BELT FOR PATIENTS WITH ABDOMINAL COLOSTOMIES

THAD. A. KROLICKI, M.D.

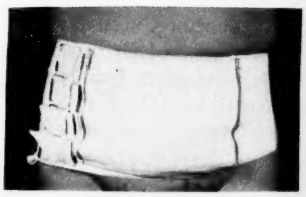
Junior Surgeon, Memorial Hospital; Surgeon, R. I. State Hospital for Mental Diseases; Associate Fellow, American Proctologic Society.



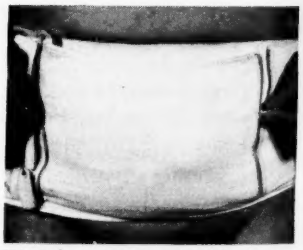
I. With perineal straps for male



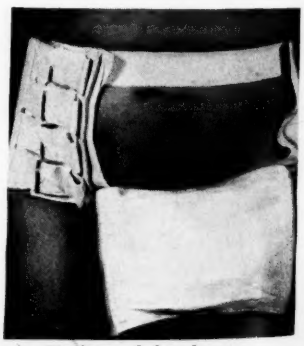
II. With pantie for female



III. Front view



IV. Fenestrum closed with zippers



V. Opened for dressing

Purpose of belt

1. To replace the conventional rubber or plastic colostomy bags.
2. To facilitate change of dressing without removing belt.
3. To give firm support to back and abdomen.

Description

Belt is adaptable to colostomies in any quadrant — the following is a belt adapted for colostomy in left lower quadrant following Miles resection of rectum. Heavy Unbleached Drilling is used for this belt, which has suitable waterproof pocket (plastic), for necessary dressing. Buckles on right side, with four heavy 1½″ straps with protected buckles to prevent tearing of clothing. The waterproof pocket can be opened to remove dressing, without taking off belt, by two strong zippers each side of pocket, the belt remaining in position, and not falling down, as it is held by connecting belt of 1½″ web at top, separate from the pocket. For men, it has two pair of flannel under-straps buckling to belt, front and back, 1″ wide. For women, two pair of flannel panty straps 2½″ wide fastened with snaps to belt.

How to Measure for Belt

This belt is 8″ deep.

1. Give exact circumference of body where upper edge of belt will come, usually 2″ above colostomy.

2. Exact circumference where lower edge of belt will be worn.

3. Exact circumference in middle of belt, that is, 4″ down from upper edge of belt. Give length of thigh straps for men and length of panty straps for women, in both cases, from lower edge of belt in front, and around perineum to lower edge of belt in back.

All measures to be taken next to flesh—standing up.
Cost of belt:

As made to above specifications is $7.50, physicians price by H. Mawby Co., Inc., 63 Washington Street, Providence, Rhode Island.
Belt may be made by any good appliance house.

NEW DIVISION OF MEDICAL EXAMINERS IN RHODE ISLAND

In accordance with the stipulations in the legislation enacted by the Rhode Island General Assembly at its recent session, the new division of medical examiners has been established within the department of justice, and under the supervision of a chief medical examiner. Dr. William H. Magill of Providence has been named chief medical examiner and for the time being he will maintain this office at 116 Waterman street where he has been in general medical practice for many years.

With the approval of the Attorney General, as required by law, Doctor Magill has appointed the County Deputies who will cover the territory or area as indicated below. It should be noted that Deputy Examiners have no jurisdiction except in the County where they reside. But regardless of the area assigned to any deputy within a given county, inability to get the man indicated would not preclude calling upon another deputy appointed for the same county. It will be the policy of the Chief Medical Examiner and of the Attorney General to cooperate fully with the members of the medical profession, and no physician should hesitate about calling in a County Deputy whenever such action seems to indicate sound judgment.

The County Deputy Medical Examiners appointed to serve under Doctor Magill, effective July 1, 1949, are:

Providence and Bristol Counties

Foster, Scituate, Glocester and Cranston
Arthur G. Randall, M.D.
North Scituate

Woonsocket, No. Smithfield, Burrillville
Edward L. Myers, M.D.
56 Winter St., Woonsocket

Pawtucket, Central Falls, Lincoln and Cumberland
Albert J. Gaudet, M.D.
592 Smithfield Ave., Pawtucket

Providence, East Providence, Johnston, No. Providence & Smithfield
John A. Picozzi, M.D.
358 Broadway, Providence
and alternately
James J. Flanagan, M.D.
36 Cliffdale Ave., Cranston

Warren, Barrington and Bristol
Ralph J. Petrucci, M.D.
88 Child St., Warren

Washington County

No. Kingstown, So. Kingstown, Narragansett and Exeter
Salvatore J. P. Turco, M.D.
170 High St., Peacedale

Charlestown, Richmond, Westerly, Hopkinton and Exeter
Frances A. Kenyon, M.D.
Woodville, R. I.

Kent County

East Greenwich, West Greenwich and Coventry
Fenwick G. Taggart, M.D.
1 Montrose St., E. Greenwich

Warwick and West Warwick
Rocco Abbate, M.D.
803 Warwick Ave., Lakewood

Newport County

Including Tiverton, Little Compton, Portsmouth, New Shoreham, Middletown, Jamestown, Newport
Philomen P. Ciarla
105 Pelham St., Newport
and alternately
Frank J. Logler, M.D.
42 Kay St., Newport

WHEN THE DIET

Needs Supplementation

Comparison of the accompanying two columns of nutritional values clearly shows why Ovaltine in milk has been so widely accepted as a highly effective *multiple dietary food supplement.*

Column A lists the National Research Council's Recommended Daily Dietary Allowances for each *100 calorie portion* in the diet of a 154-pound man of sedentary occupation. Column B lists the amounts of the same nutrients provided by a *100 calorie portion* of Ovaltine in milk.

	A N. R. C. Diet	B Ovaltine in Milk*
CALORIES	100	100
CALCIUM	40 mg.	166 mg.
IRON	0.5 mg.	1.8 mg.
PHOSPHORUS	60 mg.	139 mg.
VITAMIN A	208 I.U.	444 I.U.
THIAMINE	0.05 mg.	0.17 mg.
RIBOFLAVIN	0.08 mg.	0.30 mg.
NIACIN	0.5 mg.	1.0 mg.
ASCORBIC ACID	3.1 mg.	4.4 mg.
VITAMIN D		62 I.U.
PROTEIN	2.9 Gm.	4.7 Gm.

The easy digestibility and appealing flavor of Ovaltine in milk enhance its value as a dietary supplement. Chocolate Flavored Ovaltine is especially liked by children.

*Based on average reported values for milk. Three servings of Ovaltine, each made of ½ oz. of Ovaltine and 8 fl. oz. of whole milk, the daily dosage recommended for diet supplementation, provide 676 calories.

THE WANDER COMPANY, 360 N. MICHIGAN AVE., CHICAGO 1, ILL.

Ovaltine

Two kinds, Plain and Chocolate Flavored. Serving for serving, they are virtually identical in nutritional content.

Medical Personalities

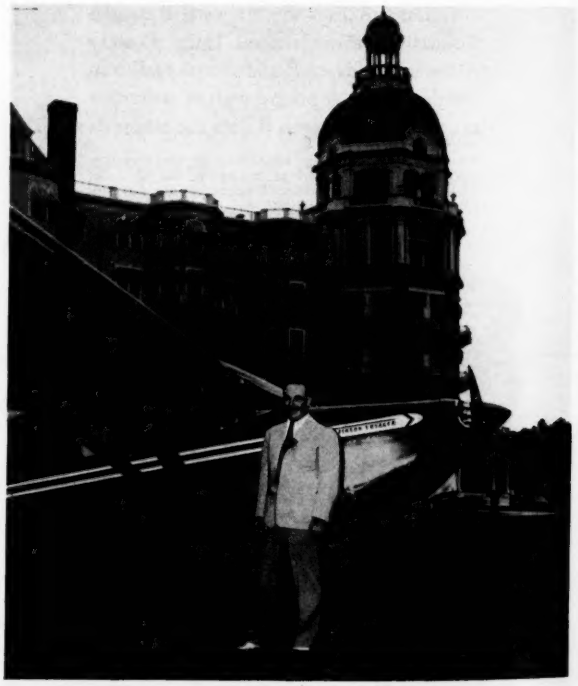
RHODE ISLAND'S FLYING SURGEON

In 1941, at the insistence of one of his patients who was a flying instructor, Dr. Francis J. King of Woonsocket learned to fly, and subsequently bought his first plane, a Taylorcraft. He had but fourteen hours of flight to his record when he was automatically grounded by reason of war regulations, since Woonsocket was within the 50 mile defense zone.

As soon as the war had ended, Doctor King renewed his flying, first using a re-conditioned Aeronca, later an Ercoupe, and now a Stinson. In 1946 he flew with Dr. Joseph Ashkins of Milford to the meeting of the College of Surgeons in Cleveland, and thus started the first of many flights to regional and national medical sessions. The following year he flew to the New York meeting, and in 1948 the cross country flight to Los Angeles was successfully negotiated, and this year the sectional meeting of the College at Buffalo found him piloting his Stinson there.

Other than attending meetings Doctor King has made three flights to Florida, one to New Orleans, and one to the tip of Nova Scotia. Professionally he has used his plane about five or six times a year to see patients at their summer homes, or to visit children at prep schools.

The photo below shows Dr. King beside his plane on the grounds of the Poland Spring House in Maine where he had flown as the delegate from the Rhode Island Medical Society to the Maine Medical Association annual meeting last June. On the day of this trip Doctor King performed an operation on a patient at the Woonsocket hospital, left the hospital at 11:15 a.m., and landed at Poland Springs just in time for lunch about 1 p.m.



Dr. Francis J. King, of Woonsocket, flying surgeon, standing beside his airplane on the grounds at the Poland Spring House in Poland Spring, Maine, where he had flown to represent the R. I. Medical Society at the annual meeting of the Maine Medical Association last June.

BOOK REVIEWS

ATLAS OF PERIPHERAL NERVE INJURIES by William R. Lyons and Barnes Woodhall. W. B. Saunders Co. 1949. $16.00

This large atlas is the effort of a neurohistologist and a neurosurgeon to depict the pathological changes in major nerves traumatized by battle forces. Their material is drawn from approximately 1000 peripheral nerve operations in the Walter Reed and Halloran General Hospitals from 1943 to 1945. It thus includes nothing about the acute phase of nerve trauma. The work also limits itself to battle wounds, and in the introduction, the authors state that "It has been considered unwise to essay some type of transition from this experience in war wounds to any present or future problem in civilian type injuries of peripheral nerves."

The volume itself is an elegant, glossy paged book with large clear type and excellent plates. The photomicrographs are so well done that a lens may be used advantageously to bring out the available detail. There are a few color plates which are more spectacular but not so informative. Preceding each group of specimens there is an explanatory text in which, although an effort is made to be simply objective, the authors' conclusions are evident.

There are 6 sections in the 329 pages, the first two being concerned with an introduction, a definition of terms and a review of normal peripheral nerve histology. The major part of the book is then devoted to the study of completely severed nerves and lesions in continuity.

There is an exhaustive display of plates showing the various changes, graduated from early to late, in the proximal and distal stumps of the completely severed nerves. A correlation is made between the gross and histological changes. A surgeon can thus decide by serial gross cuts at the operating table how much stump must be trimmed away to reach relatively unscarred nerve. It is shown, however, that in the battle type of wound the contused area is almost always more extensive than can be appreciated grossly at the time of the original injury.

There is a good discussion of the various types of incomplete nerve section including the one in which there is only temporary cessation of function without physical section. The plates, of course, are limited to examples in which there is a long standing and presumably permanent failure of return of function. There are excellent examples of the stretch lesions and compression lesions, as well as incomplete section.

The last part of the book is devoted to nerve sutures and nerve grafts. Good pathologic evidence is shown favoring, in general, nerve repair after secondary wound closure, i.e. the so-called "early" suture about 5 or 6 weeks after injury. Some lesions in continuity probably would be missed at this time. However, the pathology is usually well marked and deep, hard, scar tissue has not formed to hinder both the regenerated nerves and the surgeon. Suture materials are taken up briefly and the sad record of the various types of nerve grafts shown.

All in all, this atlas is worthy of careful study by all students of the surgery of trauma. One of its few faults is the use of long, unwieldy, clause filled sentences, which have to be read and re-read several times.

THOMAS C. MCOSKER, M.D.

CLINICAL AUSCULTATION OF THE HEART, Levine & Harvey. W. B. Saunders Company, Philadelphia, 1949.

Although this volume emphasizes the auscultatory phenomena of the heart and blood vessels in health and in disease the text is by no means limited to auscultation. The section on arrhythmias, in particular, includes general discussions of the cardiac irregularities as well as helpful suggestions in regard to therapy. The text is so profusely illustrated with phonocardiograms that one might gain the impression that these are essential for proper interpretation of auscultatory phenomena. It is not the intention of the authors to give this impression, however, but rather the method is employed in order to illustrate the various points involved in the discussions of acoustic signs. Emphasis in the book is placed chiefly upon clinical methods of diagnosis with minimum dependence upon laboratory data. There is detailed explanation of the differential between important and uninformative signs. In simple language the examiner is told how to understand as well as to interpret what he hears. There are numerous points brought

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out which are not generally recognized such as the conduction of murmurs by bony structures. Many of the subjects discussed are illustrated by brief recitals of actual cases. If it is the function of a reviewer to point out flaws in the text one is obliged to mention the fact, minor in importance, that the statement is made that auricular flutter and auricular fibrillation are due to circus movements in the auricles, theories upon which great doubt has recently been cast. The book is a useful addition to the texts dealing with the study of the heart.

CLIFTON B. LEECH

PSYCHIATRY IN GENERAL PRACTICE, by Melvin W. Thorner, M.D., D.Sc., W. B. Saunders Company, Philadelphia, London, 1948.

In a simple, non-technical fashion Dr. Melvin Thorner has written an excellent book on psychiatry. The 659 pages are crammed full of many interesting cases, each of which is followed by a discussion. In this fashion the author takes one through the realm of psychiatry, starting with people who because of minor differences or variations from normal, such as superior or inferior intelligence, find it difficult to identify with their groups and thus become maladjusted, to the more seriously disturbed individuals who cannot adjust because of serious functional or organic brain disease.

One need only look at the chapter headings to see that Dr. Thorner is presenting studies of people that are seen in every practitioner's office, regardless of the specialty. The word "people" is used advisedly because that is exactly what the author discusses. He doesn't present a case of dementia praecox but rather talks about dreamy people, nor a case of senile dementia but rather older people. This makes for easier reading and of course much greater understanding by those who have found psychiatry a difficult subject. In addition to the novel and interesting fashion of presenting case material, Dr. Thorner has a large section of his book devoted to the various approaches to therapy, including chemotherapy, physiotherapy, the shock therapies and various types of psychotherapy.

The reviewer recommends the careful reading of this book to all those who wish to learn more about a subject which is increasingly becoming more important in the everyday practice of medicine.

DAVID J. FISH, M.D.

HELP YOURSELF TO BETTER SIGHT, Margaret Darst Corbett, Prentice-Hall, Inc., New York, 1949, $2.50

In 1920 William H. Bates, a strange American exponent of the healing art, published a book, "Perfect Sight Without Glasses", in which he stated that refractive errors could be cured by certain exercises of his own devising. He at various times claimed the cure of diseases ranging from glaucoma to typhoid fever. This "system", seemingly almost forgotten, had new interest focused on it by the publication of Aldous Huxley's widely advertised book "The Art of Seeing". Huxley, with greatly reduced vision from a bilateral keratitis at the age of sixteen, had some improvement of sight towards middle life, as is common in such cases. He attributed his improvement to the Bates' method, of which he became an ardent devotee; and, in gratitude to his supposed benefactor, ventured into the field of medical literature, writing his book and other articles. The author of the present book gives her variations of the Bates' method. These consist of eye exercises described as "sunning", "palming", "nose drawing" and "brow wangling". In addition to refractive and muscular abnormalities, the author indicates that she has "cured" such conditions as congenital cataract, senile cataract, and detachment of the retina.

Established clinical and scientific observations do not support the Bates' thesis; for example, Woods in 1946, at the Johns Hopkins Hospital, after a careful study of myopic persons subjected to visual training concluded that this method was of no value.

This book may be dangerous in the hands of the impressionable who suffer from glaucoma and detachment of the retina or who are the parents of myopic children. Errors of refraction, where indicated, should be corrected by the proper ophthalmic lenses. Those selected cases of strabismus and other ocular muscular abnormalities which may be amenable to non-surgical therapy, can best be treated with curative exercises under the supervision of competent ophthalmologists. Irreparable

loss of vision may occur in organic eye diseases such as glaucoma and detachment of the retina unless they are treated without delay by the specially trained physician.

MILTON G. ROSS, M.D

MEDICAL ETYMOLOGY, by O. H. Perry Pepper, M.D., W. B. Saunders and Co., 1949.

Dr. Pepper had an interesting idea when he decided to bring out this book. We agree with him that modern young doctors ought to have a better background. A little understanding of the history of medicine and real meaning of the terms they use make them broad-minded.

It must have been a labor of love for Dr. Pepper to look up the background of all these common medical words. We are not 100 per cent with him in his objection to eponyms. In fact he is not 100 per cent himself for he puts some in.

We are sure that at times eponyms are decidedly helpful. He himself gives us "Eustachion Tube", and "Fallopian Tube". Poupart's ligament will long linger with us. Why should not an anatomical term be derived from the name of a man as well as from some object which many people know nothing about and couldn't recognize the relationship even if they did.

Take the ampulla of Vater for instance. Does the average young medical man know anything more about an ampulla than he does about the famous anatomist? All this is unimportant but we do think that Dr. Pepper made a serious mistake in arranging his names under eighteen different headings. For instance immediately after receiving the book we met the word "obturator". We were thinking of an obturator as a part of a surgical instrument. Our Secretary could not find it. Later on we discovered that she had been looking under the medical list and obturator was under the anatomical.

Ecchymosis we thought would be under surgery where we so frequently see it, and then we thought it might be under skin where it occurs. We finally find it under medicine which is certainly not where we would place it.

We see no reason why all the words shouldn't have been given in one alphabetical list as they are in the dictionary.

This is an interesting book. We trust lots of doctors will use it.

PETER PINEO CHASE, M.D.